and optic nerve as well, though not in the cortex of the five cases he examined and they did not occur in alcoholic pseudotabes. It is the same trabecular infiltration which is found in the cortex of paretics (Alzheimer[33]), but in tabes it is relatively rare, even though the pia is always thickened (Spiller[34]).

I have thought it right to mention these exceptional cases, so that the main issue should not be confused by a consideration of cases complicated by lesions of which the genesis differs so markedly from those of true tabes dorsalis. In this way the uninformed will not be deceived by criticisms of the radicular genesis of tabes; for they will know that criticisms of the nature indicated are founded upon epiphenomena which may occur in any syphilitic; although they could not be adduced with regard to the case here cited, yet they might be so adduced regarding some future case in which the cranial nerve paralysis of a tabetic was due to toxemia, arteritis, or nuclear disease. This would not be a cranial nerve tabes, for that disease, like the spinal tabes itself, is always due to the transverse radiculitis of Nageotte, whether this affects the sensory or the motor roots.

ACUTE PULMONARY ŒDEMA AS A TERMINAL EVENT IN CERTAIN FORMS OF EPILEPSY.

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At the time the observations now to be narrated were made, that is, during the period of my active connection with the Ohio Hospital for Epileptics, from 1897 to 1905, and my then close touch with the literature of epilepsy, I could find no record to indicate that the syndrome, acute pulmonary œdema, epileptic seizures, status epilepticus, and sudden death in epileptics had been recognized. My purpose was to make it the subject of a special report, and brief mention of the matter was made in several communications[1] of that period dealing more or less fully with the question of status lymphaticus and its relation to epilepsy.[2] So far as I have been able to determine, it has received no general recognition to the present time. I wish, then, to invite attention to the subject of acute œdema of the lungs as a terminal event in certain forms of epilepsy, and by the phrase "certain forms" to refer especially to that type of epilepsy in which status lymphaticus is found in morbid anatomical association.

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[33] Ein Beiträg zur Histopath. d. Tabes Dorsalis, 1905, etc.

[34] Philadelphia Neurological Society, 1908.

[1] See A Consideration of the Neuroses of Status Lymphaticus, Journal of the American Medical Association, February 13, 1904; and the article Status Lymphaticus, Reference Handbook of the Medical Sciences, p. 453.

[2] See especially the Bulletin of the Ohio Hospital for Epileptics, 1898, vol. i, No. 1; 1898, vol. i, Nos. 2 and 3; 1904, vol. ii, No. 1.

Acute œdema of the lungs, even apart from any relation to epilepsy, is an uncommonly discussed clinical event, as one may judge from a recent excellent review by Riesman[3] who notes that little or no mention of it is made in the standard treatises on the practice of medicine. Besides carefully compiling the literature on the subject, Riesman describes the history of six personally observed cases of acute pulmonary œdema, but nowhere in his study does he mention its association with epilepsy, nor with status lymphaticus. In Geysen's thorough thesis[4] devoted to sudden death in epileptics, no allusion is made to fatal pulmonary œdema nor to status lymphaticus.

The acute serous inundation of the lungs has been observed in epileptics under several types—as accompanying a single major fit with death soon succeeding; after a single attack with coma, and death in a longer interval; after a series of fits not reaching status epilepticus; and as hastening status epilepticus to a lethal termination. There is also evidence, so far as postmortem observations go, that some of the not uncommon nocturnal fatalities among epileptics (the "found-dead-in-bed" class of institutional records) in reality are instances of acute pulmonary œdema after one or more epileptic attacks, rather than of mechanical asphyxia by bedclothing or otherwise, as is commonly believed.

To illustrate the variations just alluded to, typical cases have been selected from my notes, and will be described as concisely as possible.

Case I.—The first patient was Jenny G., who was admitted to the Ohio Hospital for Epileptics in 1896, in her nineteenth year of age. Both grand mal and petit mal had existed since her seventeenth year. There were seldom more than one or two epileptic crises during a period of twenty-four hours; but a month rarely passed without at least one major attack. She was sane, and above the average epileptic inmate in intelligence, and her general health was good.

The woman had been in her usual health April 8, 1899; that evening she was in a cheerful mood, and spent some time in the attendant's room with several other inmates. At 8 P.M. she retired, and in half an hour had a severe major epileptic attack. Frothy, reddish mucus issued from her mouth during the convulsion and reappeared as it was wiped away. A comatose condition succeeded the clonic convulsions. She was seen by the physician shortly after the fit began, and was then lying on her back; the loud, stertorous breathing with occasional attempts at coughing could be heard across the large dormitory; a thin, foamy fluid escaped from the mouth and nose; at times this fluid was pinkish, and when the woman was turned on her side a gush amounting to about two ounces would follow. After several of these passive movements it was found that they only further embarrassed breathing, and she was allowed to lie on her back. There was no cessation to the appearance of the foam and the occasional gushes of fluid. The respirations during this period were sixteen to twenty a minute, and coarse rales were plainly audible at a distance from the chest. The skin was cold and clammy; the pulse of good volume and only moderately accelerated. Toward the end breathing became as short gasps, and the pulse rapid and weak. Death took place at 10 P.M., one and one-half hours after the single epileptic attack.

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[3] Acute Pulmonary Œdema, with Special Reference to a Recurrent Form, Amer. Jour. Med. Sci., January, 1907.

[4] De la mort inopinée ou rapide chez les epileptiques. Thèse No. 1114, Faculté de Médecine et de Pharmacie de Lyon, 1895.

Autopsy. The morbid anatomical findings of chief interest relate to the lungs and the anomalies of status lymphaticus. A pronounced diffuse œdema of the lungs was indicated before the removal of these viscera by the escape of voluminous gushes of thin, frothy fluid whenever the corpse was moved, and after their removal by a very heavy, boggy, waterlogged state, the free flow of bubbly serum when they were incised, and the weight (1820 grams) of both lungs. The thymus was large, fleshy, reached the level of the second rib, the seat of numerous punctate hemorrhages, 1 cm. in thickness, weighed 30 grams, and made up of closely set adenoid follicles. There was moderate enlargement of the superficial regional lymph glands, while those of the peritoneal cavity were generally large, soft, and pinkish in hue. The solitary follicles of the stomach and intestines were prominent, and so were Peyer's patches. The spleen was large (340 grams), but its follicles were not strikingly prominent. The thyroid was soft, pale, and about one-third larger than normal. There were no other visceral morbid changes.

CASE II.—Frank S., aged twenty-two years, was a victim of grand mal and beginning epileptic dementia. He was subject to periodic major attacks of much severity. The evening of March 8, 1899, he had returned to his cottage after partaking of a hearty supper in the central dining room, seemingly in his usual general good health. While standing in the main room with some companions he was suddenly seized with a fit and fell backward, forcibly striking his head on the floor. It was noticed at once that he had not breathed properly after the convulsive phase of the attack ceased, and when the physician reached the scene he found a condition of urgent dyspnœa, with frothy fluid escaping from the mouth. The coma continued, breathing became more labored, and in twenty minutes after the epileptic attack the patient expired.

Autopsy. As disclosed by the postmortem examination, a right-sided subdural hematoma with a bulk of 50 c.c., without demonstrable fracture of the bone or rupture of meningeal bloodvessels, had resulted from the fall. Foamy, blood-stained fluid covered the lips, cheeks, and nostrils, and when pressure was applied to the chest this fluid freely gushed from the mouth and nose. The lungs distended the pleural cavities, were boggy, and exuded bloody, frothy fluid during their manipulation, both before and after incision. A diffuse, reddish, œdematous infiltration appeared on section, the extent of which can be judged by the combined weight (2170 grams) of the freshly removed lungs. The thymus was quite large, bilobed, reddish in color, and of compact adenoid tissue; the tonsils were large, projecting so as almost to touch in the middle line. The superficial, bronchial, and mesenteric lymph glands were larger than ordinary. The spleen weighed 290 grams, and its follicles were large and prominent in the red pulp. The lymphoid follicles of the whole digestive tract were very prominent.

CASE III.—James B. was twenty-seven years old when death occurred. His grand mal began when fourteen or fifteen years of age; gradual dementia supervened, with periods of religious mania, between which the man was a quiet, mild, and tractable patient. There was no unusual preliminary to the several fits that resulted fatally. The patient had retired for the night, July 5, 1900, as well as usual. He had several epileptic seizures before midnight, but as nocturnal attacks were not uncommon with him, the night attendant felt no alarm. At about 3 A.M. he had a severe general convulsion, and his breathing was so difficult and peculiar that the attendant sent for the physician, upon whose arrival breathing became easier, though coarse rales could be heard from both lungs. The pulse was good at this moment. As the attendant and physician looked on, another hard fit occurred, with foamy fluid gushing from the mouth, and in this condition breathing quickly ceased and life was extinct.

Autopsy. Thin, frothy, blood-stained fluid escaped from the mouth whenever the body was moved. Both lungs were voluminous, adherent behind and laterally; œdematous fluid escaped from the incisions. They weighed 1050 grams. The thymus was thick, fleshy, with solid thymic adenoid follicles, and beset with hemorrhagic foci. The other features of status lymphaticus were enlarged and prominent intestinal and splenic follicles.

CASE IV.—Effie P., aged 20 years, sane; occasional grand mal; had been debilitated for some time. During the night of March 3, 1901, she had four severe fits; for the three succeeding days she acted queerly, was confused, but went to meals regularly. The next day a distinctly disturbed state set in, with constant incoherent talk, efforts to remove clothing and a generally dazed condition—epileptic mania. Sleep was induced that night by large doses of paraldehyde, and the following day a quiet, taciturn, sullen mental state developed. The patient went to her dinner and ate a moderate meal, but at 2. P.M. the fits again began, and by 7 P.M. her temperature was 98°, pulse 130, respirations 42; the woman was unconscious, lips cyanotic, and gurgling rales could be heard to emphasize the labored breathing. At this hour three attacks were noted by the physician, and after each one considerable frothy fluid issued from the mouth. The gurgling continued, with frothing at the mouth; two more fits were recorded before 1.30 A.M., when death occurred.

Autopsy. The findings were essentially as have been described in the preceding cases. The lungs were free in the pleural cavity, boggy and œdematous, and weighed 1230 grams. The anatomical features of status lymphaticus corresponded to those already described.

CASE V.—Arthur A., aged twenty-five years, a victim of grand mal and epileptic dementia, began to decline. Thinking the condition due to bromism, the drug was suspended. The epileptic attacks at once became more frequent, from three to five each day. About four weeks after discontinuing the bromides, a series of grand mal began toward the morning of March 11, 1901; by 10 A.M. there had been fifteen attacks. Bromides were pushed without avail. At 11 A.M. consciousness was lost and a temperature of 102° was noted; fits occurred every fifteen to thirty minutes until 5 P.M., when death ensued in the midst of a general severe convulsion which terminated the status epilepticus. Marked dyspnœa had been noted about half an hour before the end.

Autopsy. A moderate œdema of the lungs was disclosed, together with a picture characteristic of status lymphaticus.

So much for this hurried review, in which I have endeavored, by citing the salient clinical and pathological points in five typical cases, to illustrate my thesis. In passing let it be understood that the thorough investigation at autopsy precluded the existence of renal, cardiac, or cerebral disease as explaining the causation of the pulmonary œdema; and further, that in each case a negative result followed the search for foreign bodies in the air-passages, which might have induced a mechanical suffocation.

As to the relative frequency of pulmonary œdema in epileptics, the following statistics from the records of the Ohio Hospital for Epileptics will bear witness. In the years 1903, 1904, and 1905 there were a total of 203 deaths; of these, 41 followed a single epileptic attack, or were otherwise sudden, as for example, nocturnal fatalities, in which the individual was found dead in bed. In this group of 41, œdema of the lungs was noted, either as a terminal clinical event, or was verified at autopsy in 13 cases. Again, in the period above mentioned 41 patients succumbed to status epilepticus, and among these œdema of the lungs was noted in 15 cases. That the syndrome, acute œdema of the lungs and epileptic attacks is truly a "terminal event" seems evident from the foregoing recital.

The full significance of acute pulmonary œdema in epilepsy is at most conjectural, but with what my studies have disclosed as to the existence of status lymphaticus in these cases, we can suggestively recall the fact, repeatedly emphasized by those who have occupied themselves with that peculiar morbid condition, viz., that a tendency to sudden and usually fatal œdema—as of the larynx, lungs or brain—is one of the outstanding peculiarities of the victims of the lymphatic constitution. Accordingly, the observations here recorded should tend in two directions—to emphasize the importance of acute œdema of the lungs as a concurrence in epilepsy, and further to illustrate the kinship between certain types of epilepsy and status lymphaticus.

THE PHYSIOLOGICAL UTILIZATION OF SOME COMPLEX CARBOHYDRATES.

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It is still a current practice, especially among medical men, to look upon the proximate analysis of a food material as an adequate index of its nutritive value. In certain cases the data thus gained may be depended upon to give reliable information regarding food values; in others, however, the chemical statistics derived from the more familiar analytical procedures can become quite misleading. The explanation of this lies in the fact that much of the information furnished by the analyst is indirect in character. Protein is estimated on the basis of nitrogen content. This method may lead to erroneous conclusions in the case of plant products in particular, in which a diversity of nitrogenous constituents other than proteins are frequently present. The ether extract is designated as fats, although it is known frequently to contain lipoids, notably phosphatides, the real nutritive value of which is uncertain. The carbohydrates are not infrequently estimated by difference—a method which not only is subject to inherent error, but also fails to give any indication of the specific character of the substances represented.

An illustration of the illusory character of such analyses is afforded by the investigations of one of us upon mushrooms. These are highly prized in certain quarters because of a supposed richness in protein which is attested by a considerable content of nitrogen. Experimental studies have shown, however, that the latter is present in indigestible form, in part as nitrogenous celluloses; any representations of composition made on the basis of such analytical determinations as have just been reviewed are obviously misleading.